

Pathological Studies of "Sudden Death Syndrome" in Broiler Chickens

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SUMMARY

Sudden death syndrome usually occurs in heavy, fast-growing and healthy-looking broilers. Most of the affected birds are males. The characteristic necropsy changes are seen in well-fleshed broilers with edema and generalized pulmonary congestion, recently ingested feed in the crop and gizzard, distended intestine with creamy content and empty gall bladder. The liver and kidneys are slightly enlarged and the latter have patchy areas of subcapsular hemorrhage. The heart contains clotted blood in the atria but the ventricles are often empty and the left ventricle in particular assumes a hypertrophied appearance.

Microscopic examination of heart muscle reveals degeneration of fibers, separation of cardiac muscle fibers by edema and infiltration of heterophils. The lungs have severe vascular congestion, inflammatory cell infiltration in the mucosa of the secondary bronchi and edema fluid in the tertiary bronchi and interlobular connective tissue. The liver has moderate bile duct hyperplasia, periportal hepatitis and mononuclear cell infiltration adjacent to bile ducts which possibly leads to bile duct constriction. The kidneys have subcapsular and parenchymatous hemorrhage.

RÉSUMÉ

Études pathologiques du syndrome de mort subite chez le poulet de grill

Le syndrome de mort subite affecte ordinairement des poulets de grill lourds, qui profitent rapidement et qui semblent débordants de santé. Presque tous les sujets atteints sont des mâles; leur nécropsie révèle les lésions macroscopiques caractéristiques suivantes: oedème et congestion pulmonaires, aliments ingérés depuis peu, dans le jabot et le gésier, intestin rempli de matériel crémeux et vésicule biliaire vide. Le foie et les reins présentent

une légère hypertrophie; ceux-ci présentent aussi des foyers hémorragiques sous-capsulaires. Les oreillettes contiennent du sang coagulé, tandis que les ventricules sont souvent vides et que le gauche présente de l'hypertrophie.

L'examen microscopique du myocarde révèle une dégénérescence de ses fibres, lesquelles se trouvent séparées les unes des autres par de l'oedème et une infiltration d'hétérophiles. Les poumons présentent une hyperémie marquée, une infiltration de la muqueuse des bronches secondaires par des cellules inflammatoires, ainsi qu'un oedème des bronches tertiaires et du tissu conjonctif inter-lobulaire. Le foie présente une hyperplasie modérée des canaux biliaires, une hépatite péri-portale et une infiltration de la périphérie des canaux biliaires par des mononucléaires auxquels on attribue la sténose de ces canaux. Les reins présentent des hémorragies sous-capsulaires et parenchymateuses.

INTRODUCTION

Sudden death syndrome (SDS), also called "flip-over" or "heart attack", has become increasingly important as a cause of mortality in rapidly growing broiler chickens. Mortality from SDS has been reported as varying from three to 15.6% of the total mortality in affected flocks (1, 3, 4).

Studies on the pathology of the condition are limited. Cassidy *et al* (2) examined 45 specimens of broiler chickens exhibiting "flip-over" syndrome. At necropsy they found clots of blood in the heart and, using histological and histochemical methods, tried to determine if the clots occurred prior to death or postmortem. The blood clots were found in all four heart chambers and had a similar appearance in all of the birds regardless of age. The histological and histochemical examination of the clots failed to confirm them as thrombi, but rather the clots were thought to be of postmortem origin.

Other than these studies on the blood clots (2), and some on heart muscle (7), an intensive study has not been done of the other tissues in affected cases. The aim of the present study was to examine the pathological changes in several tissues and arrive at a cause of death for the syndrome. The impact of environmental factors on the etiology of SDS is described in another publication (6).

MATERIALS AND METHODS

Field cases from six affected flocks, a total of 142 birds, were found suitable for pathological studies. These were collected during various seasons of the year. Freshly dead birds or refrigerated specimens were necropsied, identified and gross lesions

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recorded. The following tissues were collected for histological examination: adrenal glands, aorta, bone, bone marrow, brain, Bursa of Fabricius, gizzard, proventriculus, gonads, heart, intestines, kidney, liver, lung, muscle, pancreas, peripheral nerves, spleen, thymus, thyroid and trachea. The tissues were fixed in ten percent phosphate-buffered formalin at room temperature before trimming. Following trimming, they were kept in a similar formalin solution before embedding in paraffin and stained with hematoxylin and eosin (5).

The technique of histological examination of the heart from each broiler was designed to give an adequate sample of all the parts of the organ. The heart was cut into six equidistant sections by saggital section; two sections from the atrium and four from the ventricle. Each section was again sectioned vertically to obtain several areas from each one. The special stains applied to heart tissue sections were van Geisen and Masson trichrome (5).

Control tissues were collected from two sources. The contact control tissues were from broiler chickens which had been raised with the group where SDS broilers were collected. Noncontact control tissues were from mycoplasma free white leghorn chickens raised in isolation at the University Research Station. In both cases, tissues taken for histopathology were similar to those collected for microscopic studies of the SDS affected birds.

RESULTS

Of the 142 broilers examined, 109 (77%) were male and all were in good flesh. The lesions found at necropsy were generally similar. The digestive tract

and particularly the crops and gizzards were full of recently ingested feed. Generalized pulmonary congestion was common in all of the birds and there was also frothy exudate in the trachea. The hearts were somewhat enlarged and the ventricles were firmly contracted. The atria were filled with blood clots. However, the ventricles in almost all cases were empty of any clots. The intestines, particularly in the duodenal area, were markedly dilated, lacked tone and the contents were pale, creamy and presented an appearance of catarrhal enteritis. The peritoneal serosa and mesentery showed marked vascular congestion and venous engorgement. The congested circumferential veins of the small intestines were outlined against the pale background of the intestine. The liver was slightly enlarged, friable and pale; the gall bladder was discolored and empty; the kidneys were greyish and pale; the thyroid gland, spleen and the thymus were all congested. The musculature of the breast and thighs appeared moist and pale.

The findings of the histopathological examinations are shown in Table I and a more detailed histopathology of lung, heart, kidney, liver and adrenals are summarized in Table II. The cilia and the surface epithelium of the trachea were intact. The mucosa of the trachea revealed mild vascular congestion, edema of the connective tissues and slight leukocytic infiltration. In 85% of the cases the histological changes in the lungs were characterized by varying degrees of vascular engorgement, by massive numbers of erythrocytes and edema of interstitial and interlobular connective tissues (Figure 1). In 64 cases the mucosa of the secondary bronchioles was infiltrated by mononuclear cells in such large numbers that they gave a

TABLE I
GENERAL HISTOPATHOLOGICAL FINDINGS IN 142 SDS BROILERS

Tissues Examined	Classification			
	Negative	Slight Degree of Involvement	Moderate Degree of Involvement	Severe Degree of Involvement
Aorta	142	0	0	0
Adrenal glands	41	75	22	4
Bone and bone marrow	142	0	0	0
Heart	43	61	27	11
Brain	142	0	0	0
Intestine	5	30	0	20
Bursa	142	0	0	0
Proventriculus	110	0	32	0
Gonads	137	3	0	2
Gizzard	142	0	0	0
Trachea	114	10	15	3
Muscle	142	0	0	0
Kidney	44	92	6	0
Pancreas	120	0	0	22
Liver	42	59	31	10
Peripheral nerve	142	0	0	0
Spleen	139	3	0	0
Lungs	22	45	50	25
Thymus	128	14	0	0
Thyroid	128	14	0	0
Parathyroid	6	56	10	0

TABLE II
DETAILED HISTOPATHOLOGICAL LESIONS OF LUNG,
KIDNEYS, ADRENALS, HEARTS AND LIVERS OF SDS
AND CONTROL BIRDS

Lesions	Groups		
	CC ^a	NCC ^b	SDS ^c
Lung: total tissues	10	60	120
Vascular congestion	0	0	120
Mononuclear cell aggregates	2	2	4
Leukocytic infiltration and inflammation of mucosa of secondary bronchiole	2	5	64
Edema of lung tissue	0	0	106
Kidneys: total tissues	10	60	99
Vascular congestion	0	0	97
Mononuclear cell aggregates	2	0	17
Hemorrhage	0	0	77
Urate crystal nephrosis	0	0	10
Adrenals: total tissues	10	60	101
Sinusoidal and vascular congestion	0	0	81
Mononuclear cell aggregates	4	0	56
Heterophilic infiltration	0	0	4
Bursal dependent nodes	3	2	8
Heart: total tissues	10	60	99
Vascular congestion	2	4	20
Myocardial degeneration	0	0	8
Mononuclear cell aggregates	2	4	71
Heterophilic infiltration	0	0	70
Bursal dependent nodes	2	6	7
Myocardial separation	0	0	50
Liver: total tissues	10	60	100
Sinusoidal congestion	0	0	52
Bile duct hyperplasia	0	0	64
Mononuclear cell aggregates	6	18	71
Heterophilic infiltration	0	0	9
Constricted bile ducts	3	2	59
Periportal hepatitis	4	0	98
Fatty degeneration	0	0	9

^aCC — contact control

^bNCC — noncontact control

^cSDS — sudden death syndrome

basophilic appearance to the area. The distension of the bronchial mucosa together with the intense leukocytic infiltration in the area surrounding the bronchi tended to occlude the lumen of the secondary bronchioles. The heart in 70% of the cases revealed degeneration of myofibers in addition to separation by edema fluid and leukocytic infiltration in the interstitium. The infiltrating cells were not all lymphocytes, some sections having polymorphonuclear leukocytes in the interstitium (Figure 2). These cells contained large eosinophilic granules in the cytoplasm and were confirmed by electron microscopy to be heterophils. The sinusoidal spaces and blood vessels of the adrenal, thyroid and parathyroid showed intense congestion. Focal lymphofollicular aggregates were common in the adrenal glands. The intestines

revealed various stages of autolysis. The thymus was congested and the kidneys showed congested vessels and subcapsular hemorrhage. The pancreas and proventriculus revealed a scattering of

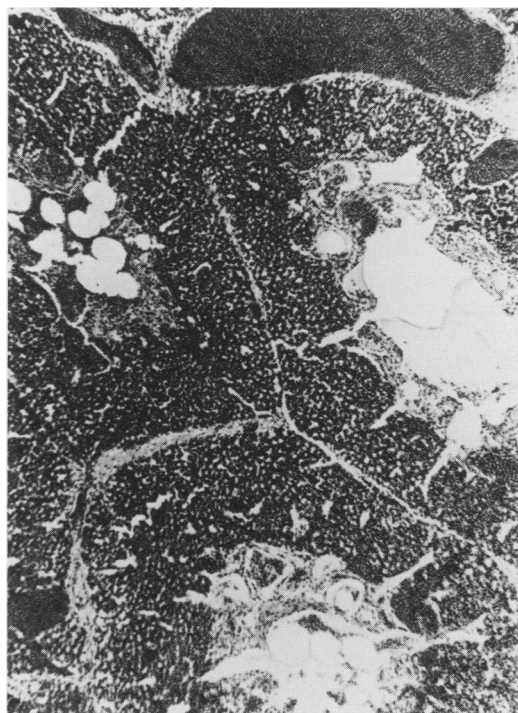


FIGURE 1. Marked pulmonary congestion.

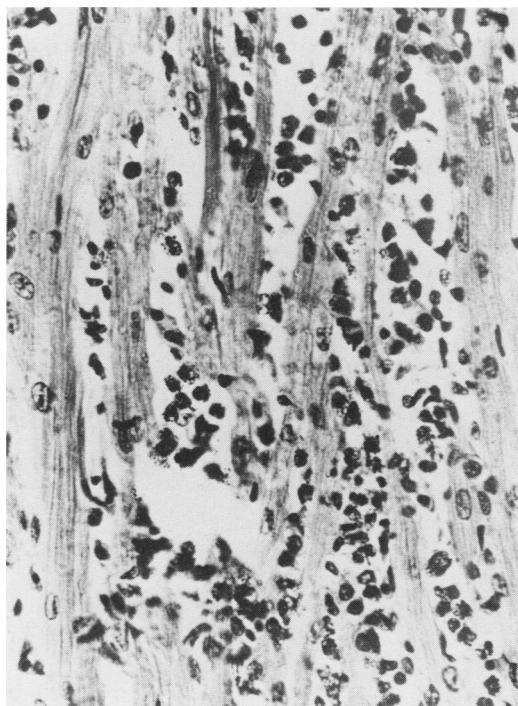


FIGURE 2. Cellular infiltration into the myocardium.

lymphofollicular aggregates as did the liver. In the liver, the portal triad areas had a deep basophilic appearance because of infiltrating cells; the presence of these cells appeared to have caused distortion and reduction of the lumen of the bile ducts.

DISCUSSION

The histological findings suggest the sequence of the pathological process in SDS. When the course of a disease is acute, most of the pathological lesions are associated with vascular disturbance. The process starts with circulatory lesions manifested by increased permeability of the peripheral circulatory system. Under the influence of physiological stresses even a healthy normal capillary may become permeable. This physiological permeability, caused by short-term increases in blood pressure, is usually reversible. However, when the stimulus surpasses the tolerance level, irreversible changes occur not only in the wall of the blood vessel, but also in the tissue which they supply. In SDS death would appear to be caused by heart damage which leads to lung edema so that the chickens are unable to breathe. Sufficient fluid is lost from the circulatory system into the lung tissue spaces to result in peripheral circulatory failure or shock. The histological changes of intense congestion and edema in the lungs result in the tissue parenchyma becoming separated from fresh blood supply therefore leading to hypoxia.

The observation of shrunken gall bladder in 100% of the cases of SDS has not been reported before. Such gall bladders may indirectly confirm field observations that the broilers dying of SDS eat normally right up to the time of death, since the physiological function of the gall bladder is to contract and supply bile rapidly to the intestine during the process of digestion.

ABSTRACT

The effects of hypaque 25% and sodium iodide in the bladder of dogs. L. Breton (Fac. vét. Med., St-Hyacinthe, Québec).

The effects of hypaque 25% and sodium iodide 10% in the bladder of dogs were studied. A group of dogs was injected with hypaque 25% and another group received sodium iodide 10%. The animals of the last group were used as controls. The following examinations were used to determine the effects: urinalysis, pneumocystography, histological examination of the bladder and necropsy. A statistical analysis of the results was performed.

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Vascular congestion is a constant feature of most of the tissues examined microscopically, particularly in the lungs where much of the effective air spaces were lost because of engorgement of pulmonary capillaries. Lymphocytic infiltration and inflammation involving the secondary bronchi and the presence of edema fluid in the alveoli considerably reduce gaseous exchange and enhance respiratory distress.

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Hypaque 25% produced very minimal signs of inflammation. These were only detectable by histological examination of the bladder wall. The urinalysis and the radiography did not show any evidence of cystitis.

Sodium iodide was found to be very irritating. It produced a severe cystitis. It was characterized by marked hematuria and proteinuria. The pneumocystogram showed thickening of the wall and an irregular outline of the internal surface of the bladder. The histological examination revealed a cystitis with necrosis, epithelial ulceration and submucosal hemorrhage.